

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	6870	epidermal adj growth adj factor	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:29			0
2	BRS	L2	205	laminin adj receptor	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:30			0
3	BRS	L3	0	((epidermal adj growth adj factor) same (modification or modified)) same ((laminin adj receptor) same (antagonist or agonist))	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:30			0
4	BRS	L4	674	(epidermal adj growth adj factor) same (modification or modified)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:34			0
5	BRS	L6	68	tyrosine adj analog	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:32			0
6	BRS	L7	262	arginine adj analog	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:32			0
7	BRS	L8	0	4 same (6 or 7)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:32			0
8	BRS	L9	0	(epidermal adj growth adj factor) same (modification or modified) same retinopathy	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:33			0
9	BRS	L10	2	(laminin adj receptor) same (antagonist or agonist) same retinopathy	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:33			0

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
10	BRS	L11	25	(epidermal adj growth adj factor) same (modification or modified) same (endothelial adj cell)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:35			0
11	BRS	L12	1	11 same (wound or wounding)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/07/14 16:36			0

=> d his

(FILE 'HOME' ENTERED AT 16:39:06 ON 14 JUL 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA'
ENTERED AT

16:39:46 ON 14 JUL 2002

L1 130542 S EPIDERMAL GROWTH FACTOR
L2 3962 S L1 (P) MODIF?
L3 1932 S (TYROSINE ANALOG) OR (ARGININE ANALOG)
L4 3935 S LAMININ RECEPTOR
L5 52 S L4 (P) (ANGONIST OR ANTAGONIST)
L6 1 S L2 (P) L5
L7 2 S L2 (P) L3
L8 2 DUPLICATE REMOVE L7 (0 DUPLICATES REMOVED)
L9 1 S L8 NOT L6
L10 68222 S RETINOPATHY
L11 0 S (ENDOTHLIAL CELL) (P) (WOUNDING OR WOUND)
L12 6 S L5 (P) L10
L13 2 DUPLICATE REMOVE L12 (4 DUPLICATES REMOVED)
L14 1 S L13 NOT (L8 OR L6)

=> log y

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9954356	A1	19991028	WO 1999-GB1211	19990421
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, RU, TJ, TM			

RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,
ES, FI, FR, GB, GR, HE, IT, LU, MC, NL, PT, SE, BF, CF, CG,
CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 9936168 A1 19991108 AU 1999-36168 19990421

EP 1073679 A1 20010207 EP 1999-918126 19990421

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, FI

PRIORITY APPLN. INFO.:

GB 1998-8407 A 19980422

WO 1999-GB1211 W 19990421

AB The present invention provides the use of natural, synthetic or
modified peptide factors derived from murine ***epidermal***
growth ***factor*** in the treatment of angiogenic diseases by
targeting ***laminin*** ***receptors***. The invention provides
agonists and ***antagonists*** which may be ***modified*** to
prevent proteolytic degradn. Use of the invention to treat retinopathy of
prematurity and promote wound healing is envisaged. The peptide factors
of the invention are based on amino acid residues 33 to 42 of murine
epidermal ***growth*** ***factor*** (mEGF). The amino
acid sequence of mEGF-(33-42) is CVIGYSGDRC. Preferred substitutions
include the use of tyrosine analogs at position 5 and arginine analogs at
position 9. Preferably the peptide factor is capped at the N terminal
with an acetyl group and at the C terminal with an amide group.
Preferably the thiol groups of cysteines are capped with acetamido Me
groups. The advantages of the invention, and the ways in which
disadvantages of previously known arrangements are overcome include: (1)
Unlike the native ***laminin*** ***receptor*** ligand
(laminin.beta.-1925_933), which is angiogenic in human models, the
mEGF33_42-derived agents are anti-angiogenic in human models, (2)
mEGF33_42 has the advantage of inhibiting both laminin- and EGF-stimulated
angiogenesis, and (3) mEGF33_42 prevents tumor cell attachment to basement
membranes.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L5 52 S L4 (P) (ANGONIST OR ANTAGONIST)
L6 1 S L2 (P) L5

=> s l2 (p) l3

L7 2 L2 (P) L3

=> duplicate remove l7

PROCESSING COMPLETED FOR L7

L8 2 DUPLICATE REMOVE L7 (0 DUPLICATES REMOVED)

=> s l8 not l6

L9 1 L8 NOT L6

=> d l9 1 ibib abs

L9 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:294219 CAPLUS
Correction of: 2001:168136

DOCUMENT NUMBER: 134:337614
Correction of: 134:233606

TITLE: Nucleic acid-based ribozyme and DNazyme modulators of
gene expression

INVENTOR(S): McSwiggen, James; Usman, Nassim; Blatt, Lawrence;
Beigelman, Leonid; Burgin, Alex; Karpeisky, Alexander;
Matulic-adamic, Jasenka; Sweedler, David; Draper,
Kenneth; Chowrira, Bharat; Stinchcomb, Dan; Beaudry,
Amber; Zinnen, Shawn; Lugwig, Janos; Sproat, Brian S.

PATENT ASSIGNEE(S): Ribozym Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 717 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001016312	A2	20010308	WO 2000-US23998	20000830
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ				
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG				
PRIORITY APPLN. INFO.:			US 1999-PV151713	19990831
			US 1999-406643	19990927
			US 1999-PV156467	19990927
			US 1999-PV156236	19990927
			US 1999-436430	19991108
			US 1999-PV169100	19991206
			US 1999-PV173612	19991229
			US 1999-474432	19991229
			US 1999-476387	19991230
			US 2000-498824	20000204
			US 2000-531025	20000320
			US 2000-PV197769	20000414
			US 2000-578223	20000523

AB Novel nucleic acid mols. useful as inhibitors of gene expression, compns., and methods for their use are provided. The invention features novel nucleic acid-based techniques (e.g., enzymic nucleic acid mols. (ribozymes), antisense nucleic acids, 2-5A antisense chimeras, triplex DNA, and antisense nucleic acids contg. RNA-cleaving chem. groups) and their use to modulate the expression of mol. targets impacting the development and progression of cancers, diabetes, obesity, Alzheimer's disease diseases, age-related diseases, and/or hepatitis B infections and related conditions. Catalytic nucleic acids were designed for site-specific cleavage of human mRNA targets encoding protein tyrosine phosphatase 1b, methionine aminopeptidase, .beta.-secretase, presenilin-1, epidermal growth factor receptor-2 (HER2/c-erb2/neu), phospholamban, telomerase, and hepatitis B virus genes. Methods for chem. synthesis of modified nucleoside triphosphates (NTPs) and RNA polymerase-catalyzed incorporation of modified NTPs into catalytic oligonucleotides are also provided. [This abstr. record os one of 6 records for this document necessitated by the large no. of index entries required to fully index the document and publication system constraints.].

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L8 2 DUPLICATE REMOVE L7 (0 DUPLICATES REMOVED)
L9 1 S L8 NOT L6

=> s retinopathy

L10 68222 RETINOPATHY

=> s (endothelial cell) (p) (wounding or wound)

4 FILES SEARCHED...

L11 0 (ENDOTHELIAL CELL) (P) (WOUNDING OR WOUND)

=> s 15 (p) l10
L12 6 L5 (P) L10

=> duplicate remove l12
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L12
L13 2 DUPLICATE REMOVE L12 (4 DUPLICATES REMOVED)

=> s l13 not (l8 or l6)
L14 1 L13 NOT (L8 OR L6)

=> d l14 1 ibib abs

L14 ANSWER 1 OF 1 MEDLINE
ACCESSION NUMBER: 2002060506 MEDLINE
DOCUMENT NUMBER: 21645779 PubMed ID: 11786424
TITLE: Synthetic peptides interacting with the 67-kd laminin
receptor can reduce retinal ischemia and inhibit
hypoxia-induced retinal neovascularization.
AUTHOR: Gebarowska Dorota; Stitt Alan W; Gardiner Thomas A;
Harriott Patrick; Greer Brett; Nelson John
CORPORATE SOURCE: Centre of Ophthalmology and Vision Science and the School
of Biology and Biochemistry, The Queen's University of
Belfast, Royal Victoria Hospital, Belfast, Northern
Ireland, United Kingdom.
SOURCE: AMERICAN JOURNAL OF PATHOLOGY, (2002 Jan) 160 (1) 307-13.
Journal code: 0370502. ISSN: 0002-9440.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020707
Entered Medline: 20020705

AB The high-affinity 67-kd ***laminin*** ***receptor*** (67LR) is
expressed by proliferating endothelial cells during retinal
neovascularization. The role of 67LR has been further examined
experimentally by administration of selective 67LR agonists and
antagonists in a murine model of proliferative ***retinopathy***
. These synthetic 67LR ligands have been previously shown to stimulate or
inhibit endothelial cell motility in vitro without any direct effect on
proliferation. In the present study, a fluorescently labeled 67LR
antagonist (EGF(33-42)) was injected intraperitoneally into mice
and its distribution in the retina was assessed by confocal scanning laser
microscopy. Within 2 hours this peptide was localized to the retinal
vasculature, including preretinal neovascular complexes, and a significant
amount had crossed the blood retinal barrier. For up to 24 hours
postinjection, the peptide was still present in the retinal vascular walls
and, to a lesser extent, in the neural retina. Non-labeled EGF(33-42)
significantly inhibited pre-retinal neovascularization in comparison to
controls treated with phosphate-buffered saline or scrambled peptide (P <
0.0001). The agonist peptide (Lam beta 1(925-933)) also significantly
inhibited proliferative ***retinopathy*** ; however, it caused a
concomitant reduction in retinal ischemia in this model by promoting
significant revascularization of the central retina (P < 0.001). Thus,
67LR appears to be an important target receptor for the modulation of
retinal neovascularization. Agonism of this receptor may be valuable in
reducing the hypoxia-stimulated release of angiogenic growth factors which
drives retinal angiogenesis.

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COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	42.61	42.82
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	-1.24	-1.24

STN INTERNATIONAL LOGOFF AT 16:46:59 ON 14 JUL 2002